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THE SPINAL CORD IN POTT'S DISEASE.[1]

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IN considering the pathology of the nervous symptoms of caries of the spine we need pay but little attention to the morbid anatomy of the bone-disease. We may define Pott's disease pathologically as an osteitis, usually tuberculous, of the bodies of one or more vertebræ, resulting in caseous necrosis and caries. Rarely the primary cause is syphilis and not tuberculosis. The bone-disease may be secondary to inflammation in neighboring structures. Finally, sometimes in septicemia secondary abscesses occur in the vertebræ. It is a curious fact, and one for which so far as I know no satisfactory explanation has been given, that this osteitis is most likely to occur in the dorsal region. The explanation sometimes offered that this region is the most exposed to injury does not explain, for the causal relation of trauma to Pott's disease has not as yet been positively proved.

The conditions mimicking Pott's disease—malig-

[1] Read at the October meeting of the Orthopedic Section of the College of Physicians, Philadelphia.

nant tumors of the vertebræ and aortic aneurism—we may dismiss without further mention.

The first question for our consideration is: Upon what do the nervous symptoms depend? Are they caused directly by the angular curvature and the consequent narrowing and angulation of the canal? In the great majority of cases, no. It is, of course, true that if a carious vertebra suddenly breaks down and fragments of bone are driven forcibly against the cord, or if the odontoid ligament gives way, we will find sudden, severe, and even fatal nervous symptoms; but ordinarily, even if the diameter of the vertebral canal be diminished by the sudden breaking down of a vertebra, the cord slips behind the projecting bone and escapes very serious compression. In by far the greater number of cases the nervous symptoms are due to inflammatory changes in the dura causing pressure upon, and later, organic changes in the cord and nerves. So soon as the osteitis has progressed far enough to cause a breaking down of the posterior surface of the body of a vertebra and ulceration of the posterior common ligament, and consequent communication with the loose tissue filling up the epidural space, inflammatory changes due to direct contiguity of tissue arise in the latter and soon appear on the external surface of the dura, ending in a pachymeningitis. When this process has progressed far enough to produce pressure upon the cord this organ, according to Hoffa,[1] becomes edematous

[1] Albert Hoffa: Transactions American Orthopedic Association. Fifth Session. Washington, 1891.

from the interference with the flow of lymph and a stasis of the fluids of its tissue. The retained lymph acts not only mechanically but also as a poison, producing at first softening of the cord with, at the same time, an increase of the connective tissue, which, when sufficient, causes sclerosis. The same is true of the peripheral nerves at their points of exit from the dura. Edema is of necessity present. As to the poisonous effect of retained lymph we need further investigation.

As the time at which communication is established between the diseased bone and the canal is, as one might say, accidental, or, rather, depends not so much upon the amount of bone-tissue diseased as upon the situation of disease in the body and the direction of its progress, it follows, as is well-known, that nervous symptoms, spasmodic abdominal pain, numbness or tingling in the extremities, alteration of the knee-jerk, paralysis, or indeed all of the symptoms referable to the cord, may precede deformity or, indeed, any sign of bone-disease. The reverse is also true. There may be severe bone-disease and marked angularity without nervous symptoms.

The liability to nervous symptoms varies with the region of the spine affected. Bouvier[1] found only five paraplegias among thirty-eight cases of lumbar caries; Coudroy de Lauréal only one paraplegia among fifty-nine similar cases. Among fifty-six cases of dorsal caries, on the contrary, Bouvier observed thirty-eight cases of paraplegia, and Coudroy nineteen cases among seventy-seven in the same

[1] Quoted by Mary Putnam Jacobi. Keating's Cyclopedia of the Diseases of Children, vol. iv, p. 646.

locality. The reason of this greater frequency of paraplegia in dorsal disease is not difficult to find. The dorsal region is the most liable to severe deformity, and to abscesses with pent-up pus, and is normally the narrowest part of the spinal canal.

The inflammation of the dura may be either simple or purulent. If simple, the external surface of the dura is reddened, with here and there patches of lymph. So long as this condition persists there are few or no severe nervous symptoms. If the inflammation be purulent, there is pus, caseous material, and organized inflammatory products. The thickening varies much, sometimes amounting to half an inch, and may surround the cord, or occupy one side, or be irregularly distributed, with nodular or even fungoid growths scattered here and there. Adhesion to the bone soon takes place. Commonly, the thickening and adhesion occur first and are greatest in the anterior portion of the dura, and hence motor cord-symptoms usually precede sensory ones. The pachymeningitis is usually confined to the neighborhood of the diseased bone, but may extend far beyond its limits. Ordinarily, the inner surface of the dura and the pia-arachnoid escape inflammatory changes till late in the disease, even though the cord is seriously disabled. This being so, we are able to say that the changes in the cord are not due to a tuberculous process in it, but to the mechanical effect of pressure and possibly the irritant effect of retained lymph, as mentioned. When the pachymeningitis invades the inner surface and the pia-arachnoid, another element is added to the destructive process.

The pressure of the thickened dura affects not only the spinal cord but also the peripheral nerves at their points of passage through it, producing Gowers' "root symptoms" and "cord symptoms." Which set of symptoms predominate will, of course, depend upon whether pressure is greatest on the nerve-roots or on the cord, and also upon which part of the periphery of the cord is most involved. As a fact, "root symptoms" vary much in intensity. There may be simply pain in the course and distribution of the affected nerves or spots of anesthesia, sometimes painful areas, muscular wasting and palsy and, curiously enough, sometimes herpes zoster. The cord symptoms, as I have said, will vary with the varying portions of the transverse area affected, and of course with the variation in the longitudinal position of the disease.

These symptoms are too well known to need repetition. In disease of the three upper cervical vertebræ, however, there is at times a curiously anomalous distribution of symptoms. Instead, as might be expected, of paralysis of all four extremities, there may be palsy of the arms, without any affection of the legs. According to Brown-Séquard the explanation is that the motor tracts for the arms are more superficial than those for the legs, and hence are earlier involved in a lesion advancing from the periphery. On the other hand, Vulpian maintains that just the opposite is true, or in other words, that in the cervical region the nerve-fibers for the arms have already entered the gray matter of the cord, while those for the legs are still in the lateral columns. He claims on this very account that the

arm-fibers will be more compressed, owing to the less density of the gray matter as compared with the white.

At autopsy in an advanced case the nerves are found much decreased in size, being sometimes mere threads. Under the microscope we find an increased amount of connective tissue, with an absence of many nerve-fibers, and sometimes with enlarged axis-cylinders. It is not strictly true to call this condition an inflammation. It is rather a degeneration. The cord ordinarily shows gross disfigurement at the seat of meningitis and curvature. It may be flattened, cylindrical, or indented. Angulation may be so marked as to persist after the removal of the cord from the canal, even in cases which at the time of death presented no symptoms. I know of no case in which there was complete severance of the cord. Shaffer[1] refers to an autopsy in which a portion of the eighth dorsal vertebra was found impinging directly upon the cord, through which it had so nearly cut its way that only a slender thread remained.

On microscopic examination in severe cases a peculiar interstitial myelitis is found. The newly formed connective tissue, which at first contains cells, later becomes changed into a dense reticulum, which much increases the hardness of the cord. The nerve-fibers are to a greater or less extent completely degenerated, though usually some are left with narrowed myelin-sheaths, and a few may be normal. The anterior part of the cord is likely to be

[1] Transactions American Orthopedic Association, vol. iv, p. 156.

most affected. The gray matter cannot be distinguished from the white, and the ganglion-cells have lost their processes and are shrunken and atrophied. Scattered everywhere are found masses of myelin, granule-corpuscles, and corpora amylacea. The walls of the small vessels are often thickened by a growth of spindle-cells arranged concentrically around the lumen, making it smaller or obliterating it entirely. This cutting off of the blood-supply of course increases the damage to the cord. The changes are greatest at the point of greatest pressure, but extend for variable distances up and down the cord. Above and below the transverse myelitis are found the usual ascending degeneration in the posterior columns and descending degeneration in the lateral pyramidal tracts. But secondary cord-changes in this disease do not always follow the usual law. According to Gowers and Charcot there may be an ascending inflammation of the lateral pyramidal tracts with consequent symptoms referable to the arms. Anomalous symptoms are also produced occasionally by inflammatory foci scattered here and there through the cord or even the medulla. Again, if the nerve-roots are affected before the cord the pressure-neuritis may invade the cord, causing streaks of sclerosis in the posterior columns. In the cases in which there is a sudden paraplegia not caused by the direct pressure of a dislocated piece of bone, hemorrhage has occurred either in the inflamed membranes or, more rarely, in the cord.

The processes by which the cord regains its functions are not altogether clear. The nerve-fibers that suffer from pressure insufficient to cause organic

change, and even those in which the myelin-sheaths are greatly narrowed, will of course resume their functions on removal of the pressure. But can a nerve-fiber in the spinal cord, if completely degenerated in a part of its course, be regenerated? In peripheral nerves regeneration certainly sometimes takes place. Quite as certainly it does not happen in the spinal cord if the destructive lesion is sudden. Apparently, however, if the lesion be a slowly increasing one, as ordinarily it is in Pott's disease, axis-cylinders may regenerate. It is also curious that there may be complete restoration of function in a cord the transverse area of which at the seat of former disease is half or less than half that of the normal.